92 History of Past Gastrointestinal Diseases

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Definition

The history of past gastrointestinal diseases encompasses disorders of the esophagus, stomach, pancreas, gall bladder, and biliary tract, as well as jaundice.

Among the esophageal diseases, *gastroesophageal reflux disease* consists of reflux of gastric contents through an incompetent lower esophageal sphincter causing symptoms. It may be associated with esophageal inflammation and, in a minority of patients, bleeding and stricture formation. An *esophageal stricture* is a fibrotic narrowing generally of its lower end as a consequence of injury, usually from reflux esophagitis. Tumors of the esophagus, both extrinsic and intrinsic, can produce narrowing of the esophageal lumen, leading to symptoms that closely mimic those of benign stricture. *Motor disorders of the esophagus* are a group of disorders characterized by motor dysfunction, some being primary esophageal diseases such as achalasia, diffuse spasm, and presbyesophagus, while others reflect esophageal manifestations secondary to other diseases.

A *peptic ulcer* is a sharply demarcated denuded area involving the mucosa, submucosa, and muscularis mucosa. It occurs commonly in the stomach and duodenum. Pain is the characteristic symptom.

Pancreatitis, inflammation of the pancreas, is classified as acute, acute recurrent (relapsing), chronic relapsing, and chronic (exocrine pancreatic insufficiency).

The most common disease of the gallbladder is associated with the presence of gallstones, with subsequent inflammation (*cholecystitis*). Cholecystitis can be acute or chronic. Stones in the common bile duct (*choledocholithisis*) or hepatic ducts may produce jaundice and/or biliary sepsis (*cholangitis*).

Jaundice (icterus) is a yellow discoloration of the skin, sclera, and mucous membranes caused by the deposition of bilirubin. It is clinically apparent when the serum bilirubin is in the range of 2 to 3 mg/dl. It results from abnormalities in bilirubin metabolism. Depending on the site of interference, hyperbilirubinemia can be unconjugated (indirect), conjugated (direct), or combined.

Technique and Clinical Significance

Esophageal Diseases

Gastroesophageal reflux disease is common. Reflux of acid-peptic or alkaline gastric contents into the lower esophagus results in stimulation of mucosal or submucosal pain receptors to produce the symptom of "heartburn" (pyrosis), which is a retrosternal burning sensation. Heartburn or esophageal pain may not only be localized to the retrosternal region but to multiple other areas such as throat, root of neck, angle of jaws, right and left anterior chest, epigastrium and right hypochondrium, and sometimes even umbilical region. Gastroesophageal reflux is a chronic condition characterized by fluctuation in symptomatology. The pain differs from duodenal ulcer pain by being more persistent and being relieved only briefly by oral antacids. The well-defined acid cycle that occurs with duodenal ulcer is not seen in this condition. Factors that reduce competence of the lower esophageal sphincter such as fatty meal, citrus juices, tomato juice, mint, chocolate, high-dose ethanol, cigarette smoking, and certain body positions such as decubitus, or sitting in certain subjects, may aggravate the heartburn. Factors that increase intra-abdominal pressure such as pregnancy or bending over, or anything that impedes the normal drainage of gastric contents such as gastric outlet obstruction, may make the symptoms worse.

Chronic reflux may result in the formation of a stricture at the lower end of the esophagus. When this occurs, the original symptoms may change to predominant dysphagia. Therefore, it is important to inquire for dysphagia in all these patients and to take a dietary history.

Dysphagia (difficulty in swallowing) is the classic symptom of the presence of *esophageal stricture*. Because gastroesophageal reflux is the most common cause for development of such a stricture, one should inquire for the presence of symptoms of reflux in the past, namely chronic recurrent heartburn. Very often, a patient's symptoms of heartburn will decrease in frequency and intensity as stricture develops. A stricture of the esophagus can also occur as a consequence of gastroesophageal surgery, inlying nasogastric tube, prolonged vomiting, and corrosive ingestion. Detailed inquiry should be made about these risk factors in the past.

Because tumors of the esophagus can cause similar symptoms and since squamous cell carcinoma is the most common tumor, one should inquire for the presence of predisposing factors in the patient, such as tylosis, corrosive ingestion and peptic esophagitis, achalasia, alcoholism, cigarette smoking, Plummer–Vinson syndrome, and past head and neck cancer.

Dysphagia, or difficulty in swallowing, is a common symptom in most patients with *motor disorders of the esophagus*. It is produced by transport problems involving the body of the esophagus related to diminished, absent, or disordered peristalsis, as well as by lower esophageal sphincter dysfunction, especially inadequate relaxation. Chest pain, other than typical heartburn, caused by esophageal distention or spasm occurs in this disease, particularly in diffuse esophageal spasm. Odynophagia, or painful swallowing, may occur as a result of swallowing initiated esophageal spasm.

Unlike organic diseases, the symptoms associated with motor disorders will have been present for a long time, generally months or years. They are also generally characterized by fluctuation, intermittency, and aggravation by emotional stress. Weight loss may be absent.

In these patients inquiry should be made about previous

esophageal and cardiac work-up as well as surgical manipulations such as bougienage or dilations or even anti-reflux or gastric surgery.

Peptic Ulcer Disease

In *duodenal ulcer*, classically, the patient complains of localized epigastric pain that occurs when the stomach is empty and is relieved by food, antacids, or vomiting. The pain may awaken the patient at night at approximately 1 to 2 A.M. It is usually not present in the morning on awakening. A characteristic feature of duodenal ulcer pain is intermittence. If the pain is constant and unchanging, the possibility of alternative diagnosis or a complication of duodenal ulcer such as penetration into the pancreas or obstruction should be considered. Vomiting is a somewhat unusual but important symptom of uncomplicated duodenal ulcer. Mild diarrhea may occur.

The pain of *gastric ulcer* is very similar to that of duodenal ulcer and the two may be indistinguishable. Sometimes gastric ulcer pain may occur a short time after eating or even during eating. The cyclical pattern of duodenal ulcer pain may be lacking. The location of the pain may be the left of the epigastrium. Vomiting may be present. Weight loss is more common than with duodenal ulcer.

Because peptic ulcer disease is a recurring condition, inquiring about past episodes is important. The pattern of exacerbations and remissions over months is called "periodicity." Very often, the patient will know the site of his previous ulcer, duodenal or gastric.

It is possible for patients with ulcer disease to have atypical upper abdominal symptoms. In this situation, careful inquiry should be made about past ulcer disease or its complications. If the symptoms elicited are suggestive of ulcer disease, the examiner must ascertain whether the patient has had any of the complications of this disease.

Hemorrhage occurs in 15 to 20% of patients with ulcer. Inquiry should be made about past episodes of black tarry stools, occult blood in the stool, anemia, or vomiting of "coffee ground" material or bright red blood, shock, or blood transfusions.

Perforations occur in 5 to 10%. This is a dramatic event that the patient will remember because he or she will in all probability have undergone surgery. Penetration is characterized by intractable pain radiating to the back, mimicking pancreatic pain. The rhythmicity of classic duodenal ulcer pain is lost. Vomiting, signs of pancreatitis, fever, and leukocytosis may be present or may have been present.

Obstruction occurs in approximately 5%. Here also, the pain may become constant and may be relieved only by vomiting. Weight loss, anorexia, and early satiety may be present.

Patients with ulcer symptoms might have undergone surgery in the past. The incidence of recurrent ulcer after surgery varies. The type of surgery undergone should be ascertained, whether it was a gastric resection, with or without a vagotomy, and the type of anastomosis.

Pancreatitis

Acute pancreatitis causes a symptom complex of abdominal pain, fever, nausea, and vomiting. It might vary from mild, vague abdominal pain to established shock with an acute abdomen. Pain is the most important feature. It usually starts acutely in the epigastrium and may bore to the back. Sitting up and leaning forward may give some relief. Progressively the pain may involve the whole abdomen and may be indistinguishable from other conditions causing an acute abdomen.

Nausea and vomiting occur frequently in association with pain. Unlike in classic gastric disorders, vomiting may not relieve this pain. Fever is another common finding. It probably results from tissue damage and does not necessarily indicate infection. However, infections, particularly of the biliary tract, should be excluded.

Shock occurs if the attack is severe. Fluid sequestration into the retroperitoneum and intestines, blood loss, and release of injury-related kinins may be the cause.

In the United States, alcohol is a major cause of acute pancreatitis. It is thus important to take a detailed history of alcohol consumption, which should include amount and duration. It takes generally 5 to 10 years of steady, heavy alcohol consumption before a person develops pancreatitis, even though the pancreas might have already sustained subclinical damage.

Biliary tract disease, namely choledocholithiasis, is an established cause of acute pancreatitis. Proposed mechanisms include stone impaction and stenosis of the sphincter of Oddi. Thus the patient is usually a middle-aged woman. The examiner should inquire about past biliary and gallbladder disease, and biliary and gallbladder surgery, and for a family history of gallbladder lithiasis and hemolytic disease.

Hypercalcemia, particularly if associated with hyperparathyroidism, is known to be associated with acute pancreatitis. The proposed pathogenetic mechanisms include intraductal lithiasis, activation of trypsin by increased concentration of calcium in pancreatic juice, and pancreatic vasculitis. Inquiry should be made for symptoms of hypercalcemia, renal lithiasis, and past diagnosis of parathyroid disease in the patient and immediate family.

The relationship between hyperlipidemia and acute pancreatitis is a clear one. Pancreatic lipase is thought to convert intrapancreatic triglycerides to toxic free fatty acids that cause inflammation of the pancreas. Lipoprotein abnormalities associated with pancreatitis are Type I (increased chylomicrons), Type IV (increased very low density lipoproteins, VLDL), and Type V (increased chylomicrons and increased VLDL). The examiner should inquire into the presence of a family history of hyperlipidemia in all young patients with acute pancreatitis.

Besides alcohol, drugs clearly associated with pancreatitis include azathioprine, estrogens, furosemide, sulfonamides, and thiazide diuretics. A number of other drugs can probably cause pancreatitis. A careful drug history is important in all patient with acute pancreatitis.

If the patient is presenting with a history of recurrent episodes of pancreatic disease, he or she might be aware of the diagnosis. In that case, direct questioning will reveal a great deal of valuable information. The following questions may be asked: Have you had any disease of the pancreas before? What did your physician call it, acute pancreatitis or chronic pancreatitis? What was thought to be causing it? What work-up was done: x-rays, ultrasound examination, CT scan? How were you treated? What was the physician's advice on discharge? Did you have any complications of pancreatitis? Was surgical intervention contemplated or done? Can records be obtained?

Chronic pancreatitis and *chronic relapsing pancreatitis* are most commonly caused by alcohol. Hyperparathyroidism, hyperlipidemia, malnutrition, and hereditary causes may also cause chronic pancreatitis. Less common etiologies include hemochromatosis, cystic fibrosis, collagen vascular diseases, and idiopathic causes. Chronic or chronic recurrent abdominal pain is the most common symptom. Malabsorption leading to weight loss may occur. Diabetes may develop. Generally, patients with chronic pancreatitis or chronic relapsing pancreatitis have had multiple hospitalizations and extensive past evaluations. Direct questions may be asked: How long have you had the pain? What work-up was done during previous episodes? Any surgery? What therapy was given?

Gallbladder and Biliary Tract Disease

Symptoms develop in about 50% of patients with *cholelithiasis*. Biliary colic is the cardinal symptom—pain that is usually constant rather than colicky, and in the upper abdomen, generally in the epigastrium or right upper quadrant. The pain may radiate to the right shoulder or back. Vomiting and fever may be present. Another common symptom of cholelithiasis is flatulent dyspepsia, a combination of upper abdominal discomfort, increased eructation, and dyspepsia. It must, however, be emphasized that flatulent dyspepsia is not a specific symptom of this disease.

The examiner should inquire about the presence of these two symptoms on an intermittent basis in these patients. Sometimes patients may appear well informed. In that case, they may be asked direct questions: Do you have any trouble with your gallbladder? Have you had an attack of gallbladder pain in the past? Have you had x-rays or ultrasound of the gallbladder? Were you jaundiced (yellow eyes and high-colored urine) when you had the last attack? Did you have fever or chills? Were you admitted in hospital? Did you require strong pain medicines to control the pain? Could reports of past evaluations be obtained from the physician and the hospital?

Choledocholithiasis may be asymptomatic. More often, a stone in the duct will produce symptoms of biliary colic described above with or without obstructive jaundice. The latter is characterized by light-colored (acholic) stools and dark-colored urine (bilirubinuria). It is thus important to inquire about the stool color in these patients.

A stone partially obstructing the common bile duct can cause ascending infection in the biliary tree called *cholangitis*, which may lead to septicemia and biliary and intrahepatic suppuration. This condition is characterized by fever, chills, jaundice, and biliary colic. Inquire if the patient had symptoms of cholangitis in the past, because it may be a recurrent disease.

Another question that must be asked of all patients with biliary tract symptoms is whether they have ever had biliary or pancreatic surgery. Postoperative biliary strictures and abscesses may produce symptoms similar to intrinsic biliary tract disease. Finally, remember that acute pancreatitis may result from the passage of biliary calculi (gallstone pancreatitis), and so a past diagnosis of "pancreatitis" may be an indication of biliary lithiasis.

Jaundice

A careful history can give the examiner a great deal of insight into the cause of jaundice. Inquiry should be made about the presence of general or systemic symptoms such as anorexia (loss or impaired appetite), weight loss, chills and fever, skin lesions such as rashes, abdominal pain, and arthritis or arthralgias.

Inquiry should be made about use of over-the-counter medications such as laxatives and sedatives. Ask about exposure to or close contact (such as sexual) with jaundiced persons and about needle sticks or parenteral drug abuse. Check if the patient has received blood transfusions or blood products. Occupational history (e.g., exposure to hepatotoxins such as carbon tetrachloride) should be looked into. Travel to a part of the world where hepatitis is endemic, such as Asia or Africa, should be considered. A prior history of jaundice may lead to the possibility of drug-induced jaundice or biliary tract disease. A family history of jaundice may lead to consideration of unconjugated hyperbilirubinemia or hemolytic syndrome, particularly if the patient is anemic (sickle cell disease, hemoglobinopathies).

If the patient consumes alcohol, obtain a quantifiable history of both recent and prior use, as well as a history of withdrawal symptoms, tolerance, and alcohol-associated illnesses such as erosive gastritis with upper gastrointestinal bleeding, pancreatitis, peripheral neuropathy, and organic brain syndrome. Recurrent episodes of jaundice in a chronic alcoholic may be due to recurrent injury, causing bouts of alcoholic hepatitis.

Rarely, viral hepatitis can be transmitted by ingestion of raw seafood. It must be remembered that viral hepatitis, particularly type B, is much more common in subjects with multiple sexual partners, such as homosexuals. Finally, a recent or remote past history of upper abdominal pain should lead the examiner to consider biliary and pancreatic disease as the possible cause for the jaundice.

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